09/392,682
Attach paper #8

********** STN Columbus **********

FILE 'HOME' ENTERED AT 13:27:34 ON 27 DEC 2000
L1 16241 HOMOLOGOUS RECOMBINATION
L2 57276 GENE THERAPY
L3 79 HOMOLOGOUS REPLACEMENT
L4 2125 3 EXON
L5 134 (MUTANT OR MUTATED)(W) EXON
L6 0 L1 (N) L5
L7 16 L1 (P) L5
L8 92895 (FANCONIS ANEMIA OR THALASSAEMIAS OR CYSTIC FIBROSIS OR SICKLE
L9 39232 (XERODERMA PIGMENTOSUM OR BLOOMS SYNDROME OR RETINOBLASTOMA
OR
L10 172 (L8 OR L9) (P) L1
L11 60 DUP REM L10 (112 DUPLICATES REMOVED)
L12 0 L11 AND L5
L13 0 L11 AND L4
L14 8 L2 AND L11

L14 ANSWER 1 OF 8 MEDLINE
TI Review article: ***gene*** ***therapy*** in gastroenterology and hepatology.
AB ***Gene*** ***therapy*** for diseases of the gastrointestinal tract is an exciting prospect because of the fundamental cure that is potentially available. The gastrointestinal system, and especially the liver, is an area that will be central to the development of ***gene*** ***therapy*** . Techniques for gene replacement include ***homologous*** ***recombination*** and gene augmentation. For the treatment of cancer antisense strategy, pro-drug activation systems and gene immunotherapy are being investigated. Gene-carrying. . . numbers of target cells in vivo is still a major barrier to clinical use. Diseases that may be helped by ***gene*** ***therapy*** include: gastrointestinal malignancies, viral hepatitis, the haemophilias, hypercholesterolaemia, alpha 1-antitrypsin deficiency, and metabolic diseases of the liver and ***cystic*** ***fibrosis*** . In this review we will outline the principles of ***gene*** ***therapy*** , delivery vectors under investigation, diseases that may benefit from this technology and some of the remaining problems to be overcome.
ACCESSION NUMBER: 1998015741 MEDLINE
DOCUMENT NUMBER: 98015741
TITLE: Review article: ***gene*** ***therapy*** in gastroenterology and hepatology.
AUTHOR: Forbes S J; Hodgson H J
CORPORATE SOURCE: Liver Group Laboratory, Royal Postgraduate Medical School, London, UK.
SOURCE: ALIMENTARY PHARMACOLOGY AND THERAPEUTICS, (1997 Oct) 11 (5) 823-36. Ref: 98
Journal code: A5D. ISSN: 0269-2813.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 199802

L14 ANSWER 2 OF 8 MEDLINE
AB In preparation for a ***gene*** ***therapy*** approach to ***cystic*** ***fibrosis*** involving the precise repair of mutations on the CF gene by ***homologous*** ***recombination*** , we developed a method that would allow for selection of the CFTR+ cells originated in gene targeting experiments on CFTR- cells in vitro. The method is based on the differential sensitivity we observed between CFTR+ and CFTR- cells to agents stimulating cyclic adenosine monophosphate (cAMP). Controlled treatment with epinephrine or forskolin allows for selectively killing CFTR- cells. The efficiency of the selection method would make it suitable for rescuing the few corrected cells originated from rare ***homologous*** ***recombination*** events.
AB In preparation for a ***gene*** ***therapy*** approach to ***cystic*** ***fibrosis*** involving the precise repair of mutations on the CF gene by ***homologous*** ***recombination*** , we developed a method that would allow for selection of the CFTR+ cells originated in gene targeting experiments on CFTR-. . . cells. The efficiency of the selection method would make it suitable for rescuing the few corrected cells originated from rare ***homologous*** ***recombination*** events.
ACCESSION NUMBER: 96050921 MEDLINE
DOCUMENT NUMBER: 96050921
TITLE: A powerful method for in vitro selection of normal versus cystic fibrosis airway epithelial cells.
AUTHOR: Vega M A; Goossens M; Besmond C
CORPORATE SOURCE: Laboratoire de Genetique Moleculaire, U.91- INSERM, Hopital H. Mondor, Creteil (Paris), France..
SOURCE: GENE THERAPY, (1994 Jan) 1 (1) 59-63.
Journal code: CCE. ISSN: 0969-7128.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199602

L14 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2000 ACS
AB A method for ***gene*** ***therapy*** of genetic or infectious disease using small fragment homologous replacement is described. The method introduces small fragments of exogenous DNA into regions of endogenous genomic DNA virtually homologous to the exogenous DNA. The exogenous DNA fragments contains sequence modification that correct mutations in the endogenous DNA or introduce mutations that alter cellular or an infecting pathogen phenotype. The method is tested to correct the .delta.F508 mutation found in exon 10 of CFTR gene in vitro in an immortalized cell line .sum.CFTE29o- derived from a ***cystic*** ***fibrosis*** patient with two .delta.F508 alleles. The defect was cor. by transfecting .sum.CFTE29o- with 491 nucleotide recA-coated CFTR ssDNA fragment contg. exon 10 and flanking introns by a no. of techniques. Allelic-specific PCR was used to assess the ***homologous*** ***recombination*** frequency. This method was also evaluated in vivo using a transgenic mouse expressing a mutant mouse CFTR gene. The same strategy was provided for the treatment of other genetic diseases including classical ***sickle*** ***cell*** ***anemia*** and ***Xeroderma*** ***pigmentosum*** . The advantage of this method is

that it can overcome the drawback of complementation technique by placing the completely repaired sequence under the control of the endogenous gene promoter so that the correct gene is expressed at appropriate levels in the cell.

TI ***Gene*** ***therapy*** of genetic or infectious diseases by small fragment homologous replacement

AB A method for ***gene*** ***therapy*** of genetic or infectious disease using small fragment homologous replacement is described. The method introduces small fragments of exogenous DNA. . . .delta.F508 mutation found in exon 10 of CFTR gene in vitro in an immortalized cell line .sum.CFTE29o- derived from a ***cystic*** ***fibrosis*** patient with two .delta.F508 alleles. The defect was cor. by transfecting .sum.CFTE29o- with 491 nucleotide recA-coated CFTR ssDNA fragment contg. exon 10 and flanking introns by a no. of techniques. Allelic-specific PCR was used to assess the ***homologous*** ***recombination*** frequency. This method was also evaluated in vivo using a transgenic mouse expressing a mutant mouse CFTR gene. The same strategy was provided for the treatment of other genetic diseases including classical ***sickle*** ***cell*** ***anemia*** and ***Xeroderma*** ***pigmentosum*** . The advantage of this method is that it can overcome the drawback of complementation technique by placing the completely repaired. . .

ACCESSION NUMBER: 2000:10571 CAPLUS
DOCUMENT NUMBER: 132:74502
TITLE: ***Gene*** ***therapy*** of genetic or infectious diseases by small fragment homologous replacement
INVENTOR(S): Gruenert, Deiter C.; Kunzelmann, Karl
PATENT ASSIGNEE(S): The Regents of the University of California, USA
SOURCE: U.S., 65 pp., Cont.-in-part of U.S. Ser. No. 409,544, abandoned.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6010908	A	20000104	US 1995-487799	19950607
US 5804383	A	19980908	US 1996-727003	19961008

PRIORITY APPLN. INFO.: US 1992-933471 19920821
US 1995-409544 19950324
US 1995-487799 19950607
REFERENCE COUNT: 20
REFERENCE(S): (4) Bertling; US 4950599 1990 CAPLUS
(6) Caplen, N; Nature Medicine 1995, V1(1) CAPLUS
(7) Cline; Pharm Ther 1985, V29, P69 CAPLUS
(9) Flotte, T; Gene Therapy 1995, V2, P29 CAPLUS
(10) Gareis, M; Cell Mol Biol 1991, V37, P191 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2000 ACS

AB The present invention relates to management and treatment of hemoglobinopathies, such as ***sickle*** ***cell*** ***anemia*** and .beta.-thalassemia. The invention also relates to developing research

animals and cell lines for the study of hemoglobinopathies and their therapies. The invention utilizes third strand oligonucleotides to target double-stranded nucleic acid sequences in or near the globin genes, or in or near sequences controlling expression of those genes to cause either a desired mutation (third-strand-targeted mutagenesis method) or nucleic acid damage to stimulate ***homologous*** ***recombination*** (third-strand-targeted ***homologous*** ***recombination*** method) with a supplied donor nucleic acid.

AB The present invention relates to management and treatment of hemoglobinopathies, such as ***sickle*** ***cell*** ***anemia*** and .beta.-thalassemia. The invention also relates to developing research animals and cell lines for the study of hemoglobinopathies and their. . . controlling expression of those genes to cause either a desired mutation (third-strand-targeted mutagenesis method) or nucleic acid damage to stimulate ***homologous*** ***recombination*** (third-strand-targeted ***homologous*** ***recombination*** method) with a supplied donor nucleic acid.

ACCESSION NUMBER: 1997:168528 CAPLUS
DOCUMENT NUMBER: 126:153644
TITLE: Treatment of hemoglobinopathies by third-strand-targeted mutagenesis or homologous recombination method
INVENTOR(S): Glazer, Peter M.
PATENT ASSIGNEE(S): Yale University, USA
SOURCE: PCT Int. Appl., 70 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9640271	A1	19961219	WO 1996-US9430	19960606
W: AU, BR, CA, CN, CZ, FI, HU, IL, JP, KP, KR, MX, NO, NZ, SG, SK, UA, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9663286	A1	19961230	AU 1996-63286	19960606

PRIORITY APPLN. INFO.: US 1995-473845 19950607
WO 1996-US9430 19960606

L14 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2000 ACS

AB The invention relates to novel human DNA sequences, targeting constructs, and methods for producing novel genes encoding thrombopoietin DNase I and .beta.-interferon by homologous recombination. The targeting constructs comprise at least: (a) a targeting sequence; (b) a regulatory sequence; (c) an exon; and (d) a splice-donor site. The targeting constructs, which can undergo homologous recombination with endogenous cellular sequences to generate a novel gene, are introduced into cells to produce homologously recombinant cells. The homologously recombinant cells are then maintained under conditions which will permit transcription of the novel gene and translation of the mRNA produced, resulting in prodn. of either thrombopoietin, DNase I, or .beta.-interferon. The invention further relates to methods of producing pharmaceutically useful prepns. contg. thrombopoietin, DNase I or .beta.-interferon from homologously recombinant cells and methods of ***gene*** ***therapy*** comprising

administering homologously recombinant cells producing thrombopoietin, DNase I, or .beta.pinterferon to a patient for therapeutic prospects.

TI Thrombopoietin, DNase I, or .beta.-interferon ***gene*** ***therapy*** , targeting sequences for ***homologous*** ***recombination*** , and treatment of platelet disorder, ***cystic*** ***fibrosis*** , or multiple sclerosis

AB . . . to methods of producing pharmaceutically useful prepns. contg. thrombopoietin, DNase I or .beta.-interferon from homologously recombinant cells and methods of ***gene*** ***therapy*** comprising administering homologously recombinant cells producing thrombopoietin, DNase I, or .beta.pinterferon to a patient for therapeutic prospects.

ACCESSION NUMBER: 1996:721777 CAPLUS
DOCUMENT NUMBER: 126:2480
TITLE: Thrombopoietin, DNase I, or .beta.-interferon ***gene*** ***therapy*** , targeting sequences for ***homologous*** ***recombination*** , and treatment of platelet disorder, ***cystic*** ***fibrosis*** , or multiple sclerosis
INVENTOR(S): Treco, Douglas A.; Heartlein, Michael W.; Hauge, Brian M.; Selden, Richard F.
PATENT ASSIGNEE(S): Transkaryotic Therapies, Inc., USA
SOURCE: PCT Int. Appl., 114 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 8
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9629411	A1	19960926	WO 1996-US3377	19960312
W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN				
US 5733746	A	19980331	US 1995-406030	19950317
AU 9653625	A1	19961008	AU 1996-53625	19960312
AU 725832	B2	20001019		
EP 815232	A1	19980107	EP 1996-910432	19960312
R: DE, FR, GB				
JP 11502122	T2	19990223	JP 1996-528475	19960312

PRIORITY APPLN. INFO.: US 1995-406030 19950317
US 1991-787840 19911105
US 1991-789188 19911105
US 1992-911533 19920710
US 1992-985586 19921203
US 1994-243391 19940513
WO 1996-US3377 19960312

L14 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2000 ACS

AB A method to correct is provided to correct the mutation(s) behind a genetic disease in the genome of somatic cells derived from an individual afflicted with this disease by contacting the cells with the corresponding non-mutant DNA-fragment to allow it to undergo ***homologous***

recombination and, thus, replace a DNA sequence of the somatic cell genome, wherein the mutation(s) is(are) located. The cells obtained can be administered to an individual as a treatment of the disease. A DNA-liposome suspension comprising the non-mutant DNA-fragment can be used as a DNA-vehicle in the process. In addn., it can be administered to an individual to obtain correction of mutation(s) by in vivo integration of the said DNA into a mutated gene by ***homologous*** ***recombination*** . Diseases for which the responsible mutations have been identified and which, thus, could be treated by the above method are autosomal and X-linked genetic disorders, such as von Willebrand's disease, ***sickle*** - ***cell*** ***anemia*** , .beta.-thalassemia, hemophilia A and B, and ***cystic*** ***fibrosis*** . Thus, lymphocytes from a patient with a homozygous nonsense mutation (R1659X/R1659X) in exon 28 of the von Willebrand factor gene were used to establish Epstein-Barr virus-transformed lymphocytes. A non-mutant DNA fragment comprised of the whole exon 28 and parts of introns 27 and 28 and corresponding to the mutant region, was amplified from normal individuals, cloned, and used to feed a culture of the above lymphocytes. Transfer of the non-mutant DNA fragment into the lymphocytes was mediated by lipofectamine, a pos. charged liposome. After the cells were fed 14 times, the repaired cells amounted roughly to 0.5% of the total tested cells.

TI ***Gene*** ***therapy*** using homologous recombination for mutation correction

AB . . . an individual afflicted with this disease by contacting the cells with the corresponding non-mutant DNA-fragment to allow it to undergo ***homologous*** ***recombination*** and, thus, replace a DNA sequence of the somatic cell genome, wherein the mutation(s) is(are) located. The cells obtained can. . . an individual to obtain correction of mutation(s) by in vivo integration of the said DNA into a mutated gene by ***homologous*** ***recombination*** . Diseases for which the responsible mutations have been identified and which, thus, could be treated by the above method are autosomal and X-linked genetic disorders, such as von Willebrand's disease, ***sickle*** - ***cell*** ***anemia*** , .beta.-thalassemia, hemophilia A and B, and ***cystic*** ***fibrosis*** . Thus, lymphocytes from a patient with a homozygous nonsense mutation (R1659X/R1659X) in exon 28 of the von Willebrand factor gene. . .

ACCESSION NUMBER: 1996:295077 CAPLUS
DOCUMENT NUMBER: 124:309568
TITLE: ***Gene*** ***therapy*** using homologous recombination for mutation correction
INVENTOR(S): Anvret, Maria; Blombaeck, Margareta; Zhang, Zhiping
PATENT ASSIGNEE(S): Swed.
SOURCE: PCT Int. Appl., 19 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9604397	A1	19960215	WO 1994-SE1038	19941103

W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, JP, KE, KG, KP, KR, KZ, LK, LT, LU, LV, MD, MG, MN,

MW, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SI, SK, TJ, TT, UA, UZ, VN
RW: KE, MW, SD, SZ, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

AU 9512854 A1 19960304 AU 1995-12854 19941103
PRIORITY APPLN. INFO.: SE 1994-2642 19940805
WO 1994-SE1038 19941103

L14 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2000 ACS

AB A compn. and method for altering the sequence of a DNA fragment by ***homologous*** ***recombination*** is described. The method may be used to correct genetic defects in mammals, e.g. those causing ***cystic*** ***fibrosis*** in man, by ***homologous*** ***recombination*** . The method is superior to complementation with cDNA because it produces a complete replacement of the defective DNA sequence and places the repaired sequence under the regulatory control of the endogenous gene promoter and so ensures that the gene is expressed at appropriate levels in the cell. An immortalized human cell line .SIGMA. CFTE29o-, derived from a ***cystic*** ***fibrosis*** patient with two .DELTA.F508 alleles, was prepd. This was transfected with recA-coated CFTR DNA contg. exon 10 and flanking introns by a no. of techniques. ***Homologous*** ***recombination*** occurred and the transfected cells expressed wild-type CFTR mRNA.

AB A compn. and method for altering the sequence of a DNA fragment by ***homologous*** ***recombination*** is described. The method may be used to correct genetic defects in mammals, e.g. those causing ***cystic*** ***fibrosis*** in man, by ***homologous*** ***recombination*** . The method is superior to complementation with cDNA because it produces a complete replacement of the defective DNA sequence and. . . the gene is expressed at appropriate levels in the cell. An immortalized human cell line .SIGMA. CFTE29o-, derived from a ***cystic*** ***fibrosis*** patient with two .DELTA.F508 alleles, was prepd. This was transfected with recA-coated CFTR DNA contg. exon 10 and flanking introns by a no. of techniques. ***Homologous*** ***recombination*** occurred and the transfected cells expressed wild-type CFTR mRNA.

ACCESSION NUMBER: 1994:291460 CAPLUS
DOCUMENT NUMBER: 120:291460
TITLE: Composition and method for altering DNA sequences by homologous recombination
INVENTOR(S): Gruenert, Dieter C.; Kunzelmann, Karl
PATENT ASSIGNEE(S): Regents of the University of California, USA
SOURCE: PCT Int. Appl., 100 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9404032	A1	19940303	WO 1993-US7917	19930820

W: AT, AU, BB, BG, BR, BY, CA, CH, CZ, DE, DK, ES, FI, GB, HU, JP, KP, KR, KZ, LK, LU, MG, MN, MW, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SK, UA, VN
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE,

BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG
EP 656747 A1 19950614 EP 1993-920268 19930820
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE
PRIORITY APPLN. INFO.: US 1992-933471 19920821
WO 1993-US7917 19930820

L14 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2000 ACS
AB A review with 105 refs. From the ***cystic*** ***fibrosis*** perspective, the discovery of the CFTR gene and the intensive research that has followed has already had a major impact. Although made somewhat complex by the large no. of different CFTR mutations detected in patients, it is now possible to readily detect between 80 and 90% of mutant alleles. However, since there is no disease-specific treatment for CF that is aimed at the basic defect, research on CFTR has led to attempts to develop molecularly based therapies. These fall into at least three general categories including ***gene*** ***therapy*** , protein replacement therapy, and designed drug therapy. Initial studies with rodents not exhibiting any disease symptoms have demonstrated the feasibility of effectively delivering expressible CFTR using a virus vehicle. The possibility of stimulating alternative Cl- channels to circumvent the CFTR block is also being explored. One of the main obstructions to the development of therapies as well as to basic CF research in general has been the lack of an animal model for the disease. Since the cloning of CFTR, several groups have been attempting to develop a "CF mouse" by interrupting the murine counterpart of CFTR using the ***homologous*** ***recombination*** -embryonic stem cell technol. This has recently been accomplished and should further accelerate progress towards completely understanding this disease.
AB A review with 105 refs. From the ***cystic*** ***fibrosis*** perspective, the discovery of the CFTR gene and the intensive research that has followed has already had a major impact.. . . on CFTR has led to attempts to develop molecularly based therapies. These fall into at least three general categories including ***gene*** ***therapy*** , protein replacement therapy, and designed drug therapy. Initial studies with rodents not exhibiting any disease symptoms have demonstrated the feasibility. . . CFTR, several groups have been attempting to develop a "CF mouse" by interrupting the murine counterpart of CFTR using the ***homologous*** ***recombination*** -embryonic stem cell technol. This has recently been accomplished and should further accelerate progress towards completely understanding this disease.
ACCESSION NUMBER: 1993:425499 CAPLUS
DOCUMENT NUMBER: 119:25499
TITLE: The cystic fibrosis transmembrane conductance regulator
AUTHOR(S): Riordan, J. R.
CORPORATE SOURCE: Res. Inst., Hosp. Sick Child., Toronto, ON, M5G 1X8, Can.
SOURCE: Annu. Rev. Physiol. (1993), 55, 609-30
CODEN: ARPHAD; ISSN: 0066-4278
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

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Attach paper # 8

	Type	L #	Hits	Search Text
1	BRS	L1	4178	homologous adj recombination
2	BRS	L6	26	homologous adj replacement
3	BRS	L11	5782	exon
4	BRS	L16	15	mutant adj exon
5	BRS	L21	1	l1 near l11
6	BRS	L26	0	l1 near l16
7	BRS	L31	0	l6 near l16
8	BRS	L36	216	l1 same l11
9	BRS	L46	0	l1 with l16
10	BRS	L51	95	"fanconi's anemia"
11	BRS	L56	244	thalassaemias
12	BRS	L61	9423	cystic fibrosis
13	BRS	L66	1340	"sickle cell anemia"
14	BRS	L71	426	"retinitis pigmentosa"
15	BRS	L81	147	"xeroderma pigmentosum"
16	BRS	L86	370	"ataxia telangiectasia"
17	BRS	L91	101	"Bloom's syndrome"
18	BRS	L96	1288	retinoblastoma
19	BRS	L101	432	"Duchennes' muscular dystrophy"
20	BRS	L106	182	"Tay-Sachs disease"
21	BRS	L41	80	l1 with l11
22	BRS	L116	0	l41 same l51
23	BRS	L121	0	l41 and l51
24	BRS	L126	0	l41 and l56
25	BRS	L136	0	41 and 51
26	BRS	L146	2	41 and 71
27	BRS	L151	3	41 and 81
28	BRS	L156	2	41 and 86
29	BRS	L161	0	41 and 91
30	BRS	L166	8	41 and 96
31	BRS	L171	2	41 and 101
32	BRS	L176	0	41 and 106
33	BRS	L131	16	41 and 61
34	BRS	L141	9	41 and 66
35	BRS	L181	7	146 or 151 or 156
36	BRS	L186	10	166 or 171